WEST Search History

DATE: Tuesday, May 13, 2003

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
L2	S-adenosylmethionine same (measur$5 or assa$4 or determin$5) and hydrolase	17	L2
L1	S-adenosylmethionine and (measur$5 or assa$4 or determin$5) and hydrolase	178	L1

END OF SEARCH HISTORY

WEST

Generate Collection | Print

Search Results - Record(s) 1 through 17 of 17 returned.

☐ 1. Document ID: US 20020133850 A1

L2: Entry 1 of 17 File: PGPB Sep 19, 2002

PGPUB-DOCUMENT-NUMBER: 20020133850
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020133850 A1

TITLE: Melon promoters for expression of transgenes in plants

PUBLICATION-DATE: September 19, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Clendennen, Stephanie K.	Portland	OR	US	
Kellogg, Jill A.	Bend	OR	US	

US-CL-CURRENT: 800/287; 536/23.6, 800/283

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 2. Document ID: US 20020123088 A1

L2: Entry 2 of 17 File: PGPB Sep 5, 2002

PGPUB-DOCUMENT-NUMBER: 20020123088
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020123088 A1

TITLE: Method of measuring total homocysteine

PUBLICATION-DATE: September 5, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Matsuyama, Naoto	Osaka		JP	
Fukuhara, Mina	Osaka		JP	
Takayama, Masaharu	Osaka		JP	
Mizuno, Koji	Kyoto		JP	

US-CL-CURRENT: 435/18; 435/25

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 3. Document ID: US 20020119507 A1

L2: Entry 3 of 17 File: PGPB Aug 29, 2002

PGPUB-DOCUMENT-NUMBER: 20020119507
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020119507 A1

TITLE: Determination method of biological component and reagent kit used therefor

PUBLICATION-DATE: August 29, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Kishimoto, Takahide	Tsuruga-shi		JP	
Sogabe, Atsushi	Tsuruga-shi		JP	
Hattori, Shizuo	Tsuruga-shi		JP	
Oka, Masanori	Tsuruga-shi		JP	
Kawamura, Yoshihisa	Tsuruga-shi		JP	

US-CL-CURRENT: 435/26

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 4. Document ID: US 20020119491 A1

L2: Entry 4 of 17 File: PGPB Aug 29, 2002

PGPUB-DOCUMENT-NUMBER: 20020119491
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020119491 A1

TITLE: High expression and production of high specific activity recombinant S-adenosyl homocysteinase (SAHH) and improved assays for S-adenosylmethionine (SAM)

PUBLICATION-DATE: August 29, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Xu, Mingxu	San Diego	CA	US	
Han, Qinghong	San Diego	CA	US	

US-CL-CURRENT: 435/7.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 20020062496 A1

L2: Entry 5 of 17 File: PGPB May 23, 2002

PGPUB-DOCUMENT-NUMBER: 20020062496
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020062496 A1

TITLE: Genes encoding p-coumarate 3-hydroxylase (C3H) and methods of use

PUBLICATION-DATE: May 23, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Chapple, Clinton C.S.	West Lafayette	IN	US	
Franke, Rochus	Kaiserslautern	IN	DE	
Ruegger, Max O.	Indianapolis		US	

US-CL-CURRENT: 800/278; 435/410, 800/284

6. Document ID: US 6335170 B1

L2: Entry 6 of 17 File: USPT Jan 1, 2002

US-PAT-NO: 6335170
DOCUMENT-IDENTIFIER: US 6335170 B1

TITLE: Gene expression in bladder tumors

DATE-ISSUED: January 1, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Orntoft; Torben F.	DK 8230 Aabyhoj			DK

US-CL-CURRENT: 435/6; 435/91.1, 435/91.2, 536/23.1, 536/24.3, 536/24.31, 536/24.33

ABSTRACT:

Methods for analyzing tumor cells, particularly bladder tumor cells employ gene expression analysis of samples. Gene expression patterns are formed and compared to reference patterns. Alternatively gene expression patterns are manipulated to exclude genes which are expressed in contaminating cell populations. Another alternative employs subtraction of the expression of genes which are expressed in contaminating cell types. These methods provide improved accuracy as well as alternative basis for analysis from diagnostic and prognostic tools currently available.

21 Claims, 24 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 15

7. Document ID: US 6306618 B1

L2: Entry 7 of 17 File: USPT Oct 23, 2001

US-PAT-NO: 6306618
DOCUMENT-IDENTIFIER: US 6306618 B1

TITLE: Homocysteine desulphurase from the protozoan trichomonas vaginalis

DATE-ISSUED: October 23, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Coombs; Graham Herbert	Glasgow			GB
Mottram; Jeremy Charles	Bearsden			GB
Pritchard; David John	Scone			GB
Campbell; Robert Stewart	Perth			GB

US-CL-CURRENT: 435/18; 435/14, 435/26, 435/4, 435/975, 530/300, 536/23.1, 536/23.2, 536/23.72

ABSTRACT:

The present invention relates to an assay for determining homocysteine, cysteine, O-acetyl-L-serine and/or methionine levels in a biological sample using an enzyme that catalyzes the degradation of homocysteine, cysteine, O-acetyl-L-serine and methionine. The enzyme being more particularly homocysteine desulphurase, a polynucleotide fragment encoding

protozoan homocysteine desulphurase, a recombinant vector comprising a polynucleotide fragment, transformed cells, the protozoan homocysteine desulphurase polypeptide, and pharmaceutical compositions comprising recombinant homocysteine desulphurase for use in medicine or veterinary medicine.

72 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 5

☐ 8. Document ID: US 6232081 B1

L2: Entry 8 of 17 File: USPT May 15, 2001

US-PAT-NO: 6232081
DOCUMENT-IDENTIFIER: US 6232081 B1

TITLE: Method for the detection of NF-.kappa.B regulatory factors

DATE-ISSUED: May 15, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Harper; Jeffrey Wade	Sugarland	TX		
Elledge; Stephen J.	Houston	TX		
Winston; Jeffrey T.	Sugar Land	TX		

US-CL-CURRENT: 435/7.1; 435/7.2, 436/501, 436/516, 436/536

ABSTRACT:

The present invention provides compositions and methods for gene identification, as well as drug discovery and assessment. In particular, the present invention provides components of an E3 complex involved in ubiquitination of cell cycle regulators and other proteins, as well as members of a class of proteins that directly function in recognition of ubiquitination targets. The present invention also provides sequences of multiple F-box proteins.

4 Claims, 33 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 15

☐ 9. Document ID: US 6117849 A

L2: Entry 9 of 17 File: USPT Sep 12, 2000

US-PAT-NO: 6117849
DOCUMENT-IDENTIFIER: US 6117849 A

TITLE: S-(+)-adenosylmethionine and 3'-azido-2', 3'-dideoxy-nucleoside complexes as potent inhibitors of HIV-replication

DATE-ISSUED: September 12, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Zimmermann; Kurt	Herborn-Seelbach			DE
Paradies; H. Heinrich	Iserlohn			DE

US-CL-CURRENT: 514/45; 514/42, 514/43, 514/46, 514/47, 514/48, 514/49, 514/50, 514/51, 514/885, 536/27.14, 536/27.31, 536/28.2

ABSTRACT:

Molecular Complexes, comprising of S-(+)-adenosylmethionine and 3'-azido-2',3'-dideoxy nucleosides are prepared, and shown to have synergistic inhibitory effects on the replication of human-immunodeficiency virus 1 & 2 in vitro and in vivo, particularly on the reverse transcriptase, and having a high therapeutic index.

23 Claims, 6 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 6

☐ 10. Document ID: US 6080549 A

L2: Entry 10 of 17 File: USPT Jun 27, 2000

US-PAT-NO: 6080549
DOCUMENT-IDENTIFIER: US 6080549 A

TITLE: Methods and materials for the diagnosis and treatment of schizophrenia and related disorders

DATE-ISSUED: June 27, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Deth; Richard C.	Waban	MA		

US-CL-CURRENT: 435/7.21; 435/15, 436/501, 436/504, 436/505, 436/63, 436/804, 436/811

ABSTRACT:

Methods for detecting schizophrenia or depression based on modifications of the contribution of the D.sub.4 receptor to phospholipid methylation levels are described herein. Individuals with schizophrenia or depression have a deficiency in phospholipid methylation activity compared with normal individuals. Methods for screening therapeutic processes or agents for use in treatment of schizophrenia or related neuropsychiatric disorders are also described.

4 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 4

☐ 11. Document ID: US 5738998 A

L2: Entry 11 of 17 File: USPT Apr 14, 1998

US-PAT-NO: 5738998
DOCUMENT-IDENTIFIER: US 5738998 A

TITLE: Compositions and methods for diagnosing schizophrenia

DATE-ISSUED: April 14, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Deth; Richard C.	Waban	MA	02168	

US-CL-CURRENT: 435/7.21; 436/161, 436/174, 436/518, 436/519, 436/811

ABSTRACT:

Methods and compositions for detecting schizophrenia based on modification of the dopamine D.sub.4 receptor by addition of an adenosyl group to methionine #313 via methionine adenosyltransferase are described herein. Individuals with schizophrenia have a deficiency in methionine adenosyltransferase activity and a lower amount of modified dopamine D.sub.4 receptor than normal individuals. Methods for screening therapeutic processes, agents and drugs for use in treatment of schizophrenia are also described.

4 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 6

☐ 12. Document ID: US 5686255 A

L2: Entry 12 of 17 File: USPT Nov 11, 1997

US-PAT-NO: 5686255
DOCUMENT-IDENTIFIER: US 5686255 A

TITLE: Compositions and methods for diagnosing schizophrenia

DATE-ISSUED: November 11, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Deth; Richard C.	Waban	MA	02168	

US-CL-CURRENT: 435/7.21; 436/161, 436/174, 436/518, 436/519, 436/811

ABSTRACT:

Methods and compositions for detecting schizophrenia based on modification of the dopamine D.sub.4 receptor by addition of an adenosyl group to methionine #313 via methionine adenosyltransferase are described herein. Individuals with schizophrenia have a deficiency in methionine adenosyltransferase activity and a lower amount of modified dopamine D.sub.4 receptor than normal individuals. Methods for screening therapeutic processes, agents and drugs for use in treatment of schizophrenia are also described.

13 Claims, 7 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 6

☐ 13. Document ID: US 5589623 A

L2: Entry 13 of 17 File: USPT Dec 31, 1996

US-PAT-NO: 5589623
DOCUMENT-IDENTIFIER: US 5589623 A

TITLE: Genetic control of ethylene biosynthesis in plants

• DATE-ISSUED: December 31, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ferro; Adolph J.	Lake Oswego	OR		
Bestwick; Richard K.	Portland	OR		
Brown; Lyle R.	Corvallis	OR		

US-CL-CURRENT: 800/283; 435/320.1, 435/469, 435/470, 800/317.3

ABSTRACT:

A method for control of ethylene biosynthesis in plants comprising a vector containing a selective gene under plant promoter control, and a DNA insert comprising codons for a functional heterologous polypeptide having AdoMetase activity and flanked by a plant promoter on one side and a polyA signal sequence on the other side; and, transforming a plant host with said vector wherein the plant host transformed thereby is capable of expressing the heterologous polypeptide having AdoMetase activity under the control of said control region. The presence of the AdoMetase gene and the expression of AdoMetase in transgenic plants lowers AdoMet levels and generates an inhibitor of ACC synthase causing a corresponding decrease in ethylene biosynthesis and precursor availability. The current construction of transgenic plants containing a copy(s) of the T3 AdoMetase gene allow for construction of plants that will control ethylene biosynthesis under restricted conditions resulting in fruits, vegetables, and flowers which have been modified internally to improve shelf life and preservation qualities.

10 Claims, 10 Drawing figures
Exemplary Claim Number: 10
Number of Drawing Sheets: 9

☐ 14. Document ID: US 5416250 A

L2: Entry 14 of 17 File: USPT May 16, 1995

US-PAT-NO: 5416250
DOCUMENT-IDENTIFIER: US 5416250 A

TITLE: Genetic control of ethylene biosynthesis in plants using S-adenosylmethionine hydrolase

DATE-ISSUED: May 16, 1995

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ferro; Adolph J.	Lake Oswego	OR		
Bestwick; Richard K.	Portland	OR		
Brown; Lyle R.	Corvallis	OR		

US-CL-CURRENT: 800/283; 435/320.1, 800/298, 800/317.3

ABSTRACT:

A method for control of ethylene biosynthesis in plants comprising a vector containing a selective gene under plant promoter control, and a DNA insert comprising codons for a functional heterologous polypeptide having AdoMetase activity and flanked by a plant promoter on one side and a polyA signal sequence on the other side; and,

transforming a plant host with said vector wherein the plant host transformed thereby is capable of expressing the heterologous polypeptide having AdoMetase activity under the control of said control region. The presence of the AdoMetase gene and the expression of AdoMetase in transgenic plants lowers AdoMet levels and generates an inhibitor of ACC synthase causing a corresponding decrease in ethylene biosynthesis and precursor availability. The current construction of transgenic plants containing a copy(s) of the T3 AdoMetase gene allow for construction of plants that will control ethylene biosynthesis under restricted conditions resulting in fruits, vegetables, and flowers which have been modified internally to improve

shelf life and preservation qualities.

10 Claims, 8 Drawing figures
Exemplary Claim Number: 1,9
Number of Drawing Sheets: 8

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw. Desc | Image

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☐ 15. Document ID: US 5366871 A

L2: Entry 15 of 17 File: USPT Nov 22, 1994

US-PAT-NO: 5366871
DOCUMENT-IDENTIFIER: US 5366871 A

TITLE: Ubiquitin-peptide extensions as enzyme substrates

DATE-ISSUED: November 22, 1994

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Rechsteiner; Martin C.	Salt Lake City	UT		
Yoo; Yung J.	Salt Lake City	UT		

US-CL-CURRENT: 435/24; 435/101, 435/105, 435/15, 435/193, 530/335

ABSTRACT:

A method for assaying for enzymes that modify peptide chains, such as protein kinases and enzymes which modify the C-terminus of the Ha-RAS protein, is defined. This is done by incubating an extract in which the enzyme being assayed for may be present contained in a reaction mixture. The reaction mixture is made up of a buffer solution, a ubiquitin peptide extension, wherein the peptide contains a sequence known to be modified by an agent in the presence of the enzyme being assayed for, and the agent known to modify the peptide extension when the enzyme is present. The incubation is stopped and the ubiquitin peptide extension is separated from the solution and analyzed for the presence of the agent modified peptide. The extent of peptide modification can be both qualitative and quantative of the enzyme being assayed for. Protein kinases can be assayed for using a ubiquitin pepide extension containing the sequence (SEQ ID NO:1), Ser-Glu-Glu-Glu-Glu-Glu in the presence of a phosphorylating agent. Farnesyl-protein transferase can be assayed for using a ubiquitin-peptide extension with the sequence: Pro-Gly-Cys-Met-Ser-Cys-Lys-Cys-Val-Leu-Ser, (SEQ ID NO:11) which are the eleven C-terminal residues of the RAS molecule, in the presence of a farnesylating agent such as .sup.3 H-farnesyl pyrophosphate. Carboxyl methyl transferase can be assayed for using a farnesylated ubiquitin-peptide extension in the presence of a methylating agent such as tritium labeled as [.sup.3 H-methyl]AdeMet.

32 Claims, 1 Drawing figures
Exemplary Claim Number: 1
Number of Drawing Sheets: 1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw. Desc | Image

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☐ 16. Document ID: US 5198346 A

L2: Entry 16 of 17 File: USPT Mar 30, 1993

US-PAT-NO: 5198346
DOCUMENT-IDENTIFIER: US 5198346 A

TITLE: Generation and selection of novel DNA-binding proteins and polypeptides

DATE-ISSUED: March 30, 1993

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Ladner; Robert C.	Ijamsville	MD		
Guterman; Sonia K.	Belmont	MA		
Kent; Rachel B.	Boxborough	MA		
Ley; Arthur C.	Newton	MA		

US-CL-CURRENT: 435/69.1; 435/252.3, 435/320.1, 435/489

ABSTRACT:

Novel DNA-binding proteins, especially repressors of gene expression, are obtained by variegation of genes encoding known binding proteins and selection for proteins binding the desired target DNA sequence. A novel selection vector may be used to reduce artifacts. Heterooligomeric proteins which bind to a target DNA sequence which need not be palindromic are obtained by a variety of methods, e.g., variegation to obtain proteins binding symmetrized forms of the half-targets and heterodimerization to obtain a protein binding the entire asymmetric target.

48 Claims, 16 Drawing figures
Exemplary Claim Number: 24
Number of Drawing Sheets: 16

☐ 17. Document ID: KR 2002065925 A WO 200151651 A2 AU 200126397 A US 20020119491 A1 EP 1250448 A2

L2: Entry 17 of 17 File: DWPI Aug 14, 2002

DERWENT-ACC-NO: 2001-451863
DERWENT-WEEK: 200309

TITLE: Assessing therapeutic levels of S-adenosylmethionine comprises measuring reaction products in sample containing glycine N-methyltransferase, (His) S-adenosyl homocysteine hydrolase and glycine

INVENTOR: HAN, Q; HOFFMAN, R M ; XU, M

PRIORITY-DATA: 2000US-176444P (January 14, 2000), 2001US-0759990 (January 12, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
KR 2002065925 A	August 14, 2002		000	C12Q001/48
WO 200151651 A2	July 19, 2001	E	028	C12Q001/48
AU 200126397 A	July 24, 2001		000	C12Q001/48
US 20020119491 A1	August 29, 2002		000	G01N033/53
EP 1250448 A2	October 23, 2002	E	000	C12Q001/48

INT-CL (IPC): C07 K 14/44; C12 N 15/52; C12 Q 1/48; G01 N 33/53

ABSTRACTED-PUB-NO: US20020119491A
BASIC-ABSTRACT:

NOVELTY - Assessing therapeutic levels of S-adenosylmethionine (SAM) in a biological fluid sample comprising measuring one or more reaction products in a sample containing glycine N-methyltransferase (GMT), an S-adenosyl homocysteine hydrolase (SAHH) or His.SAHH, and glycine, where the level of one or more products is directly proportional to the level of SAM in the sample, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a kit for assaying a sample containing SAM comprising SAHH or His.SAHH, GMT, glycine and instructions for use;

(2) an assay comprising a biological sample containing SAM, and GMT, glycine, and SAHH or His.SAHH, where SAHH or His.SAHH activity results in a product which can be measured to determine the amount of SAM in the sample;

(3) an isolated nucleic acid (sequence not given in the specification);

(4) efficient production of SAHH by expressing a cassette comprising the nucleic acid of (3);

(5) purifying His.SAHH by precipitating a suspension containing His.SAHH produced from (4), with ammonium sulfate to produce a supernatant and a precipitate, and subjecting the supernatant to His Tag recognizing affinity chromatography;

(6) purifying His.SAHH with a single chromatography step by subjecting His.SAHH from (4) to Ni-NAT affinity chromatography;

(7) measuring homocysteine in a biological fluid by contacting the fluid with His.SAHH and measuring the homocysteine to SAH conversion;

(8) a composition comprising His.SAHH which yields a single band upon analysis by sodium dodecyl sulfate (SDS) polyacrylamide gel electrophoresis;

(9) depleting excess homocysteine in a biological fluid in vivo or ex vivo by contacting the fluid with SAHH; and

Escherichia coli host cells comprising the nucleic acids.

USE - The method is useful for assaying therapeutic levels of SAM in a biological sample. The method may be used as a part of a diagnostic protocol or as part of a therapeutic protocol, where conditions or progress of the therapy may be monitored. SAHH or His.SAHH may be used as a reagent, particularly screening for inhibitors and inactivators of the enzyme for use as reagents themselves as potential therapeutics, e.g. in cancer, malaria, arthritis and other diseases. Recombinant SAHH may be used as a therapeutic cancer gene in combination with SAH analogs.
ABSTRACTED-PUB-NO:

WO 200151651A EQUIVALENT-ABSTRACTS:

NOVELTY - Assessing therapeutic levels of S-adenosylmethionine (SAM) in a biological fluid sample comprising measuring one or more reaction products in a sample containing glycine N-methyltransferase (GMT), an S-adenosyl homocysteine hydrolase (SAHH) or His.SAHH, and glycine, where the level of one or more products is directly proportional to the level of SAM in the sample, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a kit for assaying a sample containing SAM comprising SAHH or His.SAHH, GMT, glycine and instructions for use;

(2) an assay comprising a biological sample containing SAM, and GMT, glycine, and SAHH or His.SAHH, where SAHH or His.SAHH activity results in a product which can be measured to determine the amount of SAM in the sample;

(3) an isolated nucleic acid (sequence not given in the specification);

(4) efficient production of SAHH by expressing a cassette comprising the nucleic acid of (3);

(5) purifying His.SAHH by precipitating a suspension containing His.SAHH produced from (4), with ammonium sulfate to produce a supernatant and a precipitate, and subjecting the supernatant to His Tag recognizing affinity chromatography;

(6) purifying His.SAHH with a single chromatography step by subjecting His.SAHH from (4) to Ni-NAT affinity chromatography;

(7) measuring homocysteine in a biological fluid by contacting the fluid with His.SAHH and measuring the homocysteine to SAH conversion;

(8) a composition comprising His.SAHH which yields a single band upon analysis by sodium dodecyl sulfate (SDS) polyacrylamide gel electrophoresis;

(9) depleting excess homocysteine in a biological fluid in vivo or ex vivo by contacting the fluid with SAHH; and

Escherichia coli host cells comprising the nucleic acids.

USE - The method is useful for assaying therapeutic levels of SAM in a biological sample. The method may be used as a part of a diagnostic protocol or as part of a therapeutic protocol, where conditions or progress of the therapy may be monitored. SAHH or His.SAHH may be used as a reagent, particularly screening for inhibitors and inactivators of the enzyme for use as reagents themselves as potential therapeutics, e.g. in cancer, malaria, arthritis and other diseases. Recombinant SAHH may be used as a therapeutic cancer gene in combination with SAH analogs.

Terms	Documents
S-adenosylmethionine same (measur$5 or assa$4 or determin$5) and hydrolase	17

```
=> file .nash
=> s (s-adenosylmethionine) or sam
MISSING TERM AFTER THIONINE) OR
Operators must be followed by a search term, L-number, or query name.

=> s s-adenosylmethionine
L1        4702 FILE MEDLINE
L2        4996 FILE CAPLUS
L3        2985 FILE SCISEARCH
L4        1150 FILE LIFESCI
L5        3703 FILE BIOSIS
L6        3984 FILE EMBASE

TOTAL FOR ALL FILES
L7       21520 S-ADENOSYLMETHIONINE

=> s l7 and glycine N-methyltransferase and hydrolase
TOTAL FOR ALL FILES
L14          8 L7 AND GLYCINE N-METHYLTRANSFERASE AND HYDROLASE

=>  dup rem l14
PROCESSING COMPLETED FOR L14
L15          6 DUP REM L14 (2 DUPLICATES REMOVED)

=> d ibib abs 1-6

L15  ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:        2002:937303  CAPLUS
DOCUMENT NUMBER:         138:20443
TITLE:                   Endocrine disruptor screening using DNA chips of
                         endocrine disruptor-responsive genes
INVENTOR(S):             Kondo, Akihiro; Takeda, Takeshi; Mizutani, Shigetoshi;
                         Tsujimoto, Yoshimasa; Takashima, Ryokichi; Enoki,
                         Yuki; Kato, Ikunoshin
PATENT ASSIGNEE(S):      Takara Bio Inc., Japan
SOURCE:                  Jpn. Kokai Tokkyo Koho, 386 pp.
                         CODEN: JKXXAF
DOCUMENT TYPE:           Patent
LANGUAGE:                Japanese
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     JP 2002355079     A2    20021210       JP 2002-69354    20020313
PRIORITY APPLN. INFO.:                    JP 2001-73183    A  20010314
                                          JP 2001-74993    A  20010315
                                          JP 2001-102519   A  20010330
AB   A method and kit for detecting endocrine-disrupting chems. using DNA
     microarrays are claimed.  The method comprises prepg. a nucleic acid
     sample contg. mRNAs or cDNAs originating in cells, tissues, or organisms
     which have been brought into contact with a sample contg. the endocrine
     disruptor.  The nucleic acid sample is hybridized with DNA microarrays
     having genes affected by the endocrine disruptor or DNA fragments
     originating in these genes have been fixed.  The results obtained are then
     compared with the results obtained with the control sample to select the
     gene affected by the endocrine disruptor.  Genes whose expression is
     altered by tri-Bu tin, 4-octaphenol, 4-nonylphenol, di-N-Bu phthalate,
     dichlorohexyl phthalate, octachlorostyrene, benzophenone, diethylhexyl
     phthalate, diethylstilbestrol (DES), and 17-.beta. estradiol (E2), were
     found in mice by DNA chip anal.

L15  ANSWER 2 OF 6  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:        2002:43345  CAPLUS
DOCUMENT NUMBER:         136:319709
TITLE:                   Transcriptional profiling reveals global defects in
                         energy metabolism, lipoprotein, and bile acid
                         synthesis and transport with reversal by leptin
                         treatment in Ob/ob mouse liver
AUTHOR(S):               Liang, Chien-Ping; Tall, Alan R.
```

CORPORATE SOURCE: Division of Molecular Medicine, Department of Medicine, Columbia University, New York, NY, 10032, USA
SOURCE: Journal of Biological Chemistry (2001), 276(52), 49066-49076
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Leptin, a hormone secreted by adipose tissue, has been shown to have a major influence on hepatic lipid and lipoprotein metab. To characterize changes in lipid and lipoprotein gene expression in mouse liver, suppression subtractive hybridization and cDNA microarray anal. were used to identify mRNAs differentially expressed after leptin treatment of ob/ob mice. Ob/ob mice showed a profound decrease in mRNAs encoding genes controlling bile acid synthesis and transport as well as a variety of apolipoprotein genes and hepatic lipase with reversal upon leptin administration, suggesting that leptin coordinately regulates high d. lipoprotein and bile salt metab. Leptin administration also resulted in decreased expression of genes involved in fatty acid and cholesterol synthesis, glycolysis, gluconeogenesis, and urea synthesis, and increased expression of genes mediating fatty acid oxidn., ATP synthesis, and oxidant defenses. The changes in mRNA expression are consistent with a switch in energy metab. from glucose utilization and fatty acid synthesis to fatty acid oxidn. and increased respiration. The latter changes may produce oxidant stress, explaining the unexpected finding that leptin induces a battery of genes involved in antioxidant defenses. Expression cluster anal. revealed responses of several sets of genes that were kinetically linked. Thus, the mRNA levels of genes involved in fatty acid and cholesterol synthesis are rapidly (<1 h) repressed by leptin administration, in assocn. with an acute decrease in plasma insulin levels and decreased sterol regulator element-binding protein-1 expression. In contrast, genes participating in fatty acid oxidn. and ketogenesis were induced more slowly (24 h), following an increase in expression of their common regulatory factor, peroxisome proliferator-activated receptor .alpha.. However, the regulation of genes involved in high d. lipoprotein and bile salt metab. shows complex kinetics and is likely to be mediated by novel transcription factors.

REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:683289 CAPLUS
DOCUMENT NUMBER: 135:340385
TITLE: Quantitative proteomic analysis of mouse liver response to the peroxisome proliferator diethylhexylphthalate (DEHP)
AUTHOR(S): MacDonald, Neil; Chevalier, Stephan; Tonge, Robert; Davison, Matthew; Rowlinson, Rachel; Young, Janice; Rayner, Steve; Roberts, Ruth
CORPORATE SOURCE: Syngenta Central Toxicology Laboratory, Cheshire, Alderley Park, Macclesfield, SK10 4TJ, UK
SOURCE: Archives of Toxicology (2001), 75(7), 415-424
CODEN: ARTODN; ISSN: 0340-5761
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Peroxisome proliferators (PPs) are a diverse group of chems. that cause hepatic proliferation, suppression of apoptosis, peroxisome proliferation and liver tumors in rodents. The biochem. response to PPs involves changes in the expression of peroxisomal .beta.-oxidn. enzymes and fatty acid transport proteins such as acyl-CoA oxidase and liver fatty acid binding protein. The response to PPs is mediated by the peroxisome proliferator-activated receptor .alpha. (PPAR.alpha.) and the livers of PPAR.alpha.-null transgenic mice do not develop tumors in response to PPs. In order to identify the mol. pathways underlying the adverse effects of PPs in rodent liver, we carried out two-dimensional differential gel electrophoresis to provide quant. proteomic analyses of diethylhexylphthalate (DEHP)-treated wild-type or PPAR.alpha.-null mouse livers. Since tumorigenesis is both PP- and PPAR.alpha.-dependent,

analyses were focused on these changes. Fifty-nine proteins were identified where altered expression was both PPAR.alpha.- and PP-dependent. In addn., six proteins regulated by the deletion of PPAR.alpha. were identified, possibly indicating an adaptive change in response to the loss of this receptor. The proteins that we identified as being regulated by PPAR.alpha. are known to be involved in lipid metab. pathways, but also in amino acid and carbohydrate metab., mitochondrial bioenergetics and in stress responses including several genes not previously reported to be regulated by PPAR.alpha.. These data provide novel insights into the pathways utilized by PPs and may assist in the identification of early markers rodent nongenotoxic hepatocarcinogenesis.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 97:694435 SCISEARCH
THE GENUINE ARTICLE: XV706
TITLE: Pancreatic exocrine secretion is blocked by inhibitors of methylation
AUTHOR: Capdevila A; DechaUmphai W; Song K H; Borchardt R T; Wagner C (Reprint)
CORPORATE SOURCE: VANDERBILT UNIV, SCH MED, DEPT BIOCHEM, 620 LIGHT HALL, NASHVILLE, TN 37232 (Reprint); VANDERBILT UNIV, SCH MED, DEPT BIOCHEM, NASHVILLE, TN 37232; DEPT VET AFFAIRS MED CTR, NASHVILLE, TN 37212; UNIV KANSAS, DEPT PHARMACEUT CHEM, SIMON RES LABS, LAWRENCE, KS 66047
COUNTRY OF AUTHOR: USA
SOURCE: ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (1 SEP 1997) Vol. 345, No. 1, pp. 47-55.
Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0003-9861.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 46
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A number of early experiments suggested a relationship between methyl group metabolism and the exocrine secretion of the pancreas. These included nutritional studies showing that ethionine, the ethyl analog of methionine which inhibits cellular methylation reactions, is a specific pancreatic toxin. Other studies indicated that protein carboxymethylation might be involved. We now show that in vivo ethionine inhibits amylase secretion from freshly isolated rat pancreatic acini, while in vitro ethionine inhibits amylase secretion from the ARA2J pancreatic cell line. S-Adenosylhomocysteine (SAH) is a product inhibitor of all methyltransferase reactions involving **S-adenosylmethionine** (SAM), and treatments that elevate cellular levels of SAH such as inhibition of S-adenosylhomocysteine **hydrolase** and the in vitro addition of adenosine and homocysteine result in the inhibition of amylase secretion in both isolated pancreatic acini and AR42J cells. Measurement of SAM and SAH levels in AR42J cells shows that inhibition of secretion is more closely related to elevation of SAH levels than to a decrease in the SAM/SAH ratio. Small G-proteins are carboxymethylated on the C-terminal prenylated cysteine and inhibitors of membrane-associated prenylcysteine methyltransferase, N-acetylfarnesylcysteine, N-acetylgeranylgeranylcysteine, and farnesylthioacetic acid (FTA), block secretion in AR42J cells. N-Acetylgeranylcysteine is not an inhibitor of the methyltransferase and does not inhibit amylase secretion. FTA inhibits membrane-associated prenylcysteine methyltransferase from AR42J cells with a K-i in the 45-69 mu M range. These results suggest that a methylation event is needed for pancreatic exocrine secretion which may be the reversible methylation of a G-protein involved in signal transduction or membrane trafficking. (C) 1997 Academic Press.

L15 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1995:918737 CAPLUS
DOCUMENT NUMBER: 123:333343
TITLE: Specific staining of **glycine N-methyltransferase**

AUTHOR(S): Santos, Fatima; Amorim, Antonio; Koempf, Jost
CORPORATE SOURCE: Faculdade de Ciencias, Univ. Porto, Oporto, Port.
SOURCE: Electrophoresis (1995), 16(10), 1898-9
CODEN: ELCTDN; ISSN: 0173-0835
PUBLISHER: VCH
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Glycine N-methyltransferase** from rabbit, human, rat and pig livers was sepd. by isoelec. focusing and a specific functional staining method was developed through the detection of sarcosine produced from the methylation of glycine. Isoenzyme patterns obtained in the various species tested differ both in the no. of bands and apparent isoelec. points. These differences may explain the contradictory data on the subunit structure and glycosylation status of the enzyme reported so far.

L15 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1
ACCESSION NUMBER: 1978:19513 CAPLUS
DOCUMENT NUMBER: 88:19513
TITLE: Tissue distribution of **S-adenosylmethionine** and S-adenosylhomocysteine in rat. Effect of age, sex and methionine administration on the metabolism of **S-adenosylmethionine**, S-adenosylhomocysteine and polyamines
AUTHOR(S): Eloranta, Terho O.
CORPORATE SOURCE: Dep. Biochem., Univ. Kuopio, Kuopio, Finland
SOURCE: Biochemical Journal (1977), 166(3), 521-9
CODEN: BIJOAK; ISSN: 0006-2936
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The distribution of **S-adenosylmethionine** (I), S-adenosylhomocysteine (II), methionine adenosyltransferase (EC 2.5.1.6) (III), and S-adenosylhomocysteine **hydrolase** (EC 3.3.1.1) (IV) in rat tissues was similar in males and females and changed only slightly with age. The sp. activity of IV was greater than that of III, and the concn. of I was greater than that of II in all tissues. However, the hepatic I/II ratio depended on food supply and age. Methionine administration (i.p.) produced a transient increase in hepatic I and II concns. and brain I was elevated during the first 2 h after methionine injection. Simultaneous glycine administration decreased the rise in I concn. induced by methionine. The tissue concn. of methionine may be the rate-limiting factor in I formation. **Glycine N-methyltransferase** may have a regulatory role in hepatic utilization of I.

=> s s-adenosylmethionine and depression
TOTAL FOR ALL FILES
L22 505 S-ADENOSYLMETHIONINE AND DEPRESSION

=> s l22 and (assay or measur? or quant? or determin?)
TOTAL FOR ALL FILES
L29 81 L22 AND (ASSAY OR MEASUR? OR QUANT? OR DETERMIN?)

=> s l29 and hydrolase
TOTAL FOR ALL FILES
L36 4 L29 AND HYDROLASE

=> dup rem l36
PROCESSING COMPLETED FOR L36
L37 2 DUP REM L36 (2 DUPLICATES REMOVED)

=> d ibib abs

L37 ANSWER 1 OF 2 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 83230847 MEDLINE
DOCUMENT NUMBER: 83230847 PubMed ID: 6860362
TITLE: **Depression** of human sperm motility by inhibition of enzymatic methylation.
AUTHOR: Sastry B V; Janson V E

CONTRACT NUMBER: AG-02077 (NIA)
HD-10607 (NICHD)
SOURCE: BIOCHEMICAL PHARMACOLOGY, (1983 Apr 15) 32 (8) 1423-32.
Journal code: 0101032. ISSN: 0006-2952.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198307
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19830708

AB Alteration of membrane fluidity during enzymatic methylation of membrane phosphatidyl-ethanolamine (PE) and neutralization of negative charges of membrane proteins due to methylation of carboxyl groups may contribute to sperm motility. Therefore, enzymatic phospholipid methylation and carboxymethylation, and the consequences of their inhibition on motility, were studied using human sperm. These studies gave the following results. Human sperm homoganates contained two phospholipid N-methyltransferases (PMT) which converted PE to phosphatidylcholine (PC) in the presence of **S-adenosylmethionine** (SAM). The first PMT converted PE to phosphatidyl-N-methylethanolamine (PME). In had a Km of 4.0 microM and a pH optimum of 8.0. The second PMT converted PME to phosphatidyl-N,N-dimethylethanolamine and PC. It had a Km of 71 microM and a pH optimum of 10.0. Spermatozoa also contained protein carboxymethylase (PCM) and methyl aceptor protein (MAP). The intracellular levels of S-adenosylhomocysteine (SAH), an inhibitor of SAM-mediated methylations, were increased by adding adenosine (100 microM), L-homocysteine thiolactone (L-HCT, 10 microM), and erythro-9-(2-hydroxy-3-nonyl)-adenine (EHNA, 10 microM), an inhibitor of adenosine deaminase, to human sperm ejaculates that had been diluted with sodium phosphate buffer at pH 7.4 and 25 degrees. The motility index of each sperm suspension was **determined** every hour for 4 hr. In the presence of the mixture of adenosine, L-HCT and EHNA, the motility index was depressed by 57%. Under similar conditions, phospholipid methylation was depressed by 48%. Similar experiments were also conducted in the presence of 3-deazaadenosine (Deaza, 80 microM), a selective inhibitor of SAH **hydrolase**. In the presence of adenosine and L-HCT, Deaza depressed the motility index by 60% and phospholipid methylation by 86%. The potencies of SAH in the inhibition of phospholipid methylation and protein carboxymethylation in sperm homogenates had the following order: PMT I greater than PCM greater than PMT II. These observations indicate that the PMT system and/or the PCM-MAP system play a significant role in the regulation of human sperm motility.

=> d ibib abs 2

L37 ANSWER 2 OF 2 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 83157824 EMBASE
DOCUMENT NUMBER: 1983157824
TITLE: **Depression** of human sperm motility by inhibition of enzymatic methylation.
AUTHOR: Rama Sastry B.V.; Janson V.E.
CORPORATE SOURCE: Dep. Pharmacol., Vanderbilt Univ. Sch. Med., Nashville, TN 37232, United States
SOURCE: Biochemical Pharmacology, (1983) 32/8 (1423-1432).
CODEN: BCPCA6
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
029 Clinical Biochemistry
028 Urology and Nephrology
LANGUAGE: English

AB Alteration of membrane fluidity during enzymatic methylation of membrane phosphatidylethanolamine (PE) and neutralization of negative charges of membrane proteins due to methylation of carboxyl groups may contribute to sperm motility. Therefore, enzymatic phospholipid methylation and carboxymethylation, and the consequences of their inhibition on motility, were studied using human sperm. These studies gave the following results.

Human sperm homogenates contained two phospholipid N-methyltransferases (PMT) which converted PE to phosphatidylcholine (PC) in the presence of **S-adenosylmethionine** (SAM). The first PMT converted PE to phosphatidyl-N-methylethanolamine (PME). It had a K(m) of 4.0 .mu.M and a pH optimum of 8.0. The second PMT converted PME to phosphatidyl-N,N-dimethylethanolamine and PC. It had a K(m) of 71 .mu.M and a pH optimum of 10.0. Spermatozoa also contained protein carboxymethylase (PCM) and methyl acceptor protein (MAP). The intracellular levels of S-adenosylhomocysteine (SAH), an inhibitor of SAM-mediated methylations, were increased by adding adenosine (100 .mu.M), L-homocysteine thiolactone (L-HCT, 10 .mu.M), and erythro-9-(2-hydroxy-3-nonyl)-adenine (EHNA, 10 .mu.M), an inhibitor of adenosine deaminase, to human sperm ejaculates that had been diluted with sodium phosphate buffer at pH 7.4 and 25.degree.. The motility index of each sperm suspension was **determined** every hour for 4 hr. In the presence of the mixture of adenosine, L-HCT and EHNA, the motility index was depressed by 57%. Under similar conditions, phospholipid methylation was depressed by 48%. Similar experiments were also conducted in the presence of 3-deazaadenosine (Deaza, 80 .mu.M), a selective inhibitor of SAH **hydrolase**. In the presence of adenosine and L-HCT, Deaza depressed the motility index by 60% and phospholipid methylation by 86%. The potencies of SAH in the inhibition of phospholipid methylation and protein carboxymethylation in sperm homogenates had the following order: PMT I > PCM > PMT II. These observations indicate that the PMT system and/or the PCM-MAP system play a significant role in the regulation of human sperm motility.

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=> s  l29 and transferase

TOTAL FOR ALL FILES
L44          3 L29 AND TRANSFERASE
=> dup rem l44
PROCESSING COMPLETED FOR L44
L45           3 DUP REM L44 (0 DUPLICATES REMOVED)

=> d ibib abs 1-3

L45  ANSWER 1 OF 3     MEDLINE
ACCESSION NUMBER:    88304607     MEDLINE
DOCUMENT NUMBER:     88304607   PubMed ID: 3406427
TITLE:               Clinical correlations of one-carbon metabolism
                     abnormalities.
AUTHOR:              Tolbert L C; Monti J A; Walter-Ryan W; Alarcon R D; Bahar
                     B; Keriotis J T; Allison J G; Cates A; Antun F; Smythies J
                     R
CORPORATE SOURCE:    Department of Psychiatry, University of Alabama,
                     Birmingham.
SOURCE:              PROGRESS IN NEURO-PSYCHOPHARMACOLOGY AND BIOLOGICAL
                     PSYCHIATRY, (1988) 12 (4) 491-502.
                     Journal code: 8211617. ISSN: 0278-5846.
PUB. COUNTRY:        ENGLAND: United Kingdom
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         198809
ENTRY DATE:          Entered STN: 19900308
                     Last Updated on STN: 19900308
                     Entered Medline: 19880921
AB   1. Ninety psychiatric inpatients with a DSM III diagnosis of
     schizophrenia, mania, or major depression were studied. 2. Upon
     admission/transfer to the Clinical Studies Unit, and prior to discharge,
     measurements of symptom severity (BPRS, Ham-D, Young's Mania
     Scale) and blood samples were obtained. 3. Erythrocytes from these paired
     (admission and discharge) blood samples were assayed for methionine
     adenosyltransferase (MAT) activity and phosphatidylcholine (PC) content.
     4. Comparisons were made between the changes in MAT Vmax, or % PC, and
     changes in symptom severity. 5. For the majority of the patients (79.3%
     of the schizophrenics; 84.6% of the depressives; and 93.8% of the manics),
     clinical improvement was associated with a "normalization" of enzyme
     activity. The association between changes in % PC and clinical response
     did not achieve significant correlation.
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L45 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1988:402555 CAPLUS
DOCUMENT NUMBER: 109:2555
TITLE: Decreased calcium-binding and calcium-ATPase activities in heart sarcolemma upon phospholipid methylation
AUTHOR(S): Panagia, Vincenzo; Elimban, Vijayan; Ganguly, Pallab K.; Dhalla, Naranjan S.
CORPORATE SOURCE: Fac. Med., Univ. Manitoba, Winnipeg, MB, R3E 0W3, Can.
SOURCE: Molecular and Cellular Biochemistry (1987), 78(1), 65-71
CODEN: MCBIB8; ISSN: 0300-8177
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Heart sarcolemma has been shown to possess 3 catalytic sites (I, II and III) for Me **transferase** activity (Panagia, V., et al., 1984). The effect of phosphatidylethanolamine N-methylation on ATP-independent Ca2+ binding and ATPase activities in isolated rat heart sarcolemma was examd. Both low-affinity (1.25 mM Ca2+) and high-affinity (50 .mu.M Ca2+) Ca2+-binding activities decreased following incubation of sarcolemmal membranes with **S-adenosylmethionine** (AdoMet) under optimal conditions for site II and III. Similarly, Ca2+-ATPase activities **measured** at 1.25 mM and 4 mM Ca2+ were depressed by phospholipid N-methylation. S-Adenosylhomocysteine, a specific inhibitor of phospholipid N-methylation, prevented the **depression** of low-affinity Ca2+ binding and Ca2+-ATPase activities, whereas the methylation-induced effect on the high-affinity Ca2+ binding was not influenced by this agent. Pretreatment of sarcolemma with Me acetimidate-HCl, an amino group-blocking agent, also prevented the methylation-induced inhibition of both Ca2+ binding and Ca2+-ATPase. A further decrease in Ca2+ binding and Ca2+-ATPase activities together with a marked increase in the intramembranal level of PC was seen when membranes were methylated under the site III conditions in the presence of phosphatidyldimethylethanolamine as exogenous substrate. There was no effect of phospholipid methylation on sarcolemmal Na+-K+-ATPase and Mg2+-ATPase activities. These results indicate a role of phospholipid N-methylation in the regulation of sarcolemmal Ca2+-ATPase and low-affinity ATP-independent Ca2+ binding.

L45 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1984:214497 BIOSIS
DOCUMENT NUMBER: BA77:47481
TITLE: **DEPRESSION** OF HUMAN SPERM MOTILITY BY INHIBITION OF ENZYMATIC METHYLATION.
AUTHOR(S): SASTRY B V R; JANSON V E
CORPORATE SOURCE: DEP. PHARMACOL., VANDERBILT UNIV. SCH. MED., NASHVILLE, TENN. 37232.
SOURCE: BIOCHEM PHARMACOL, (1983) 32 (8), 1423-1432.
CODEN: BCPCA6. ISSN: 0006-2952.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Alteration of membrane fluidity during enzymatic methylation of membrane phosphatidylethanolamine (PE) and neutralization of negative charges of membrane proteins due to methylation of carboxyl groups may contribute to sperm motility. Enzymatic phospholipid methylation and carboxylation, and the consequences of their inhibition on motility, were studied using human sperm. These studies gave the following results. Human sperm homoganates contained 2 phospholipid N-methyltransferases (PMT) which converted PE to phosphatidylcholine (PC) in the presence of **S-adenosylmethionine** (SAM). The 1st PMT converted PE to phosphatidyl-N-methylethanolamine (PME). It had a Km of 4.0 .mu.M and a pH optimum of 8.0. The 2nd PMT converted PME to phosphatidyl-N,N-dimethylethanolamine and PC. It had a Km of 71 .mu.M and a pH optimum of 10.0. Spermatozoa also contained protein carboxymethylase (PCM) and methyl acceptor protein (MAP). The intracellular levels of S-adenosylhomocysteine (SAH), an inhibitor of SAM-mediated methylations, were increased by adding adenosine (100 .mu.M), L-homocysteine thiolactone (L-HCT, 10 .mu.M) and erythro-9-(2-hydroxy-3-nonyl)-adenine (EHNA, 10 .mu.M), an inhibitor of adenosine deaminase, to human sperm ejaculates that were diluted with Na2PO4 buffer at pH 7.4 and 25.degree.. The motility index of each sperm

suspension was **determined** every hour for 4 h. In the presence of the mixture of adenosine, L-HCT and EHNA, the motility index was depressed by 57%. Under similar conditions, phospholipid methylation was depressed by 48%. Similar experiments were conducted in the presence of 3-deazaadenosine (Deaza, 80 .mu.M), a selective inhibitor of SAH hydrolase. In the presence of adenosine and L-HCT, Deaza depressed the motility index by 60% and phospholipid methylation by 86%. The potencies of SAH in the inhibition of phospholipid methylation and protein carboxymethylation in sperm homogenates had the following order: PMT I > PCM > PMT II. The PMT system and/or the PCM-MAP system evidently play a significant role in the regulation of human sperm motility.

FILE 'HOME' ENTERED AT 11:22:59 ON 13 MAY 2003

=> file .Nas
=> s (S-adenosylmethionine) and (measur? or determin? or anal? or quantif?)
L1 2510 FILE MEDLINE
L2 1968 FILE CAPLUS
L3 1143 FILE SCISEARCH
L4 483 FILE LIFESCI
L5 1553 FILE BIOSIS
L6 1750 FILE EMBASE

TOTAL FOR ALL FILES
L7 9407 (S-ADENOSYLMETHIONINE) AND (MEASUR? OR DETERMIN? OR ANAL? OR QUANTIF?)

=> d ab

L7 ANSWER 1 OF 9407 MEDLINE
AB Rickettsia prowazekii, the causative agent of epidemic typhus, is an obligate, intracellular, parasitic bacterium that grows within the cytoplasm of eucaryotic host cells. Rickettsiae exploit this intracellular environment by using transport systems for the compounds available in the host cell's cytoplasm. **Analysis** of the R. prowazekii Madrid E genome sequence revealed the presence of a mutation in the rickettsial metK gene, the gene encoding the enzyme responsible for the synthesis of **S-adenosylmethionine** (AdoMet). Since AdoMet is required for rickettsial processes, the apparent inability of this strain to synthesize AdoMet suggested the presence of a rickettsial AdoMet transporter. We have confirmed the presence of an AdoMet transporter in the rickettsiae which, to our knowledge, is the first bacterial AdoMet transporter identified. The influx of AdoMet into rickettsiae was a saturable process with a K(T) of 2.3 micro M. Transport was inhibited by S-adenosylethionine and S-adenosylhomocysteine but not by sinfungin or methionine. Transport was also inhibited by 2,4-dinitrophenol, suggesting an energy-linked transport mechanism, and by N-ethylmaleimide. AdoMet transporters with similar properties were also identified in the Breinl strain of R. prowazekii and in Rickettsia typhi. By screening Escherichia coli clone banks for AdoMet transport, the R. prowazekii gene coding for a transporter, RP076 (sam), was identified. AdoMet transport in E. coli containing the R. prowazekii sam gene exhibited kinetics similar to that seen in rickettsiae. The existence of a rickettsial transporter for AdoMet raises intriguing questions concerning the evolutionary relationship between the synthesis and transport of this essential metabolite.

=> s l1 and methyltransferase
L8 961 FILE MEDLINE
L9 609 FILE CAPLUS
L10 308 FILE SCISEARCH
L11 172 FILE LIFESCI
L12 429 FILE BIOSIS
L13 636 FILE EMBASE

TOTAL FOR ALL FILES
L14 3115 L1 AND METHYLTRANSFERASE

=> s l17 and homocysteine hydrolase
L17 NOT FOUND

The L-number entered could not be found. To see the definition of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s l14 and homocysteine hydrolase
L15 1 FILE MEDLINE
L16 4 FILE CAPLUS
L17 2 FILE SCISEARCH
L18 0 FILE LIFESCI
L19 0 FILE BIOSIS
L20 1 FILE EMBASE

TOTAL FOR ALL FILES
L21 8 L14 AND HOMOCYSTEINE HYDROLASE

=> dup rem
ENTER L# LIST OR (END):l21
PROCESSING COMPLETED FOR L21
L22 6 DUP REM L21 (2 DUPLICATES REMOVED)

=> d ibib abs

L22 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:596348 CAPLUS
DOCUMENT NUMBER: 137:292851
TITLE: Altered levels of **S-adenosylmethionine** and S-adenosylhomocysteine in the brains of L-isoaspartyl (D-aspartyl) O-**methyltransferase**-deficient mice
AUTHOR(S): Farrar, Christine; Clarke, Steven
CORPORATE SOURCE: Department of Chemistry and Biochemistry and the Molecular Biology Institute, UCLA, Los Angeles, CA, 90095-1569, USA
SOURCE: Journal of Biological Chemistry (2002), 277(31), 27856-27863
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB L-Isoaspartyl (D-aspartyl) O-**methyltransferase** (PCMT1) is a protein repair enzyme that initiates the conversion of abnormal D-aspartyl and L-isoaspartyl residues to the normal L-aspartyl form. In the course of this reaction, PCMT1 converts the Me donor **S-adenosylmethionine** (AdoMet) to S-adenosylhomocysteine (AdoHcy). Due to the high level of activity of this enzyme, particularly in the brain, it seemed of interest to investigate whether the lack of PCMT1 activity might alter the concns. of these small mols. AdoMet and AdoHcy were **measured** in mice lacking PCMT1 (Pcmt1-/-), as well as in their heterozygous (Pcmt1+/-) and wild type (Pcmt1+/+) littermates. Higher levels of AdoMet and lower levels of AdoHcy were found in the brains of Pcmt1-/- mice, and to a lesser extent in Pcmt1+/- mice, when compared with Pcmt1+/+ mice. In addn., these levels appear to be most significantly altered in the hippocampus of the Pcmt1-/- mice. The changes in the AdoMet/AdoHcy ratio could not be attributed to increases in the activities of methionine adenosyltransferase II or S-adenosylhomocysteine hydrolase in the brain tissue of these mice. Because changes in the AdoMet/AdoHcy ratio could potentially alter the overall excitatory state of the brain, this effect may play a role in the progressive epilepsy seen in the Pcmt1-/- mice.
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 2-6

L22 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:763235 CAPLUS
DOCUMENT NUMBER: 135:314399
TITLE: Detection of variations in the DNA methylation profile of genes in the **determining** the risk of disease

INVENTOR(S): Berlin, Kurt; Piepenbrock, Christian; Olek, Alexander
PATENT ASSIGNEE(S): Epigenomics A.-G., Germany
SOURCE: PCT Int. Appl., 636 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 68
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001077373	A2	20011018	WO 2001-DE1486	20010406
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
DE 10019058	A1	20011220	DE 2000-10019058	20000406
WO 2001077373	A2	20011018	WO 2001-XA1486	20010406
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, CF, CG, CI, CM, GA, GW, ML, MR, NE, SN, TD, TG				
WO 2001077373	A2	20011018	WO 2001-XB1486	20010406
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, CF, CG, CI, CM, GA, GW, ML, MR, NE, SN, TD, TG				
EP 1274865	A2	20030115	EP 2001-953936	20010406
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
EP 1278892	A1	20030129	EP 2001-940158	20010406
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.:
DE 2000-10019058 A 20000406
DE 2000-10019173 A 20000407
DE 2000-10032529 A 20000630
DE 2000-10043826 A 20000901
WO 2001-DE1486 W 20010406
WO 2001-EP3969 W 20010406

AB The invention relates to an oligonucleotide kit as probe for the detection of relevant variations in the DNA methylation of a target group of genes. The invention further relates to the use of the same for **detg.** the gene variant with regard to DNA methylation, a medical device, using an oligonucleotide kit, a method for **detg.** the methylation state of an individual and a method for the establishment of a model for establishing the probability of onset of a disease state in an individual.

Such diseases may be: undesired pharmaceutical side-effects; cancerous diseases; CNS dysfunctions, injuries or diseases; aggressive symptoms or relational disturbances; clin., psychol. and social consequences of brain injury; psychotic disorders and personality disorders; dementia and/or assocd. syndromes; cardiovascular disease, dysfunction and damage; dysfunction, damage or disease of the gastrointestinal tract; dysfunction, damage or disease of the respiratory system; injury, inflammation, infection, immunity and/or anastasis; dysfunction, damage or disease of the body as an abnormal development process; dysfunction, damage or disease of the skin, muscle, connective tissue or bones; endocrine and

metabolic dysfunction, damage or disease; headaches or sexual dysfunction. This abstr. record is one of several records for this document necessitated by the large no. of index entries required to fully index the document and publication system constraints.

L22 ANSWER 3 OF 6 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 97:785240 SCISEARCH
THE GENUINE ARTICLE: YB619
TITLE: Biosynthesis and accumulation of D-ononitol in Vigna umbellata in response to drought stress
AUTHOR: Wanek W (Reprint); Richter A
CORPORATE SOURCE: UNIV VIENNA, INST PLANT PHYSIOL, POB 285, A-1091 VIENNA, AUSTRIA (Reprint)
COUNTRY OF AUTHOR: AUSTRIA
SOURCE: PHYSIOLOGIA PLANTARUM, (OCT 1997) Vol. 101, No. 2, pp. 416-424.
Publisher: MUNKSGAARD INT PUBL LTD, 35 NORRE SOGADE, PO BOX 2148, DK-1016 COPENHAGEN, DENMARK.
ISSN: 0031-9317.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; AGRI
LANGUAGE: English
REFERENCE COUNT: 45

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Metabolic responses to water deficit that lead to an accumulation of cyclitols, have been examined in rice bean (Vigna umbellata [Thunb.] Ohwi et Ohashi). Imposition of drought stress by withholding water from the soil for 9 days led to an accumulation of D-ononitol (1D-4-O-methyl-myo-inositol) which was most pronounced in leaves (from 33 to 88 mu mol g(-1) dry mass). However, the activity of the enzyme myo-inositol 6-O-**methyltransferase** (m60MT, EC 2.1.1.X), which catalyzes the synthesis of ononitol from myo-inositol and S-adenosyl-L-methionine (AdoMet). increased in stems but not in leaves during the drought stress experiment. Detailed **analysis** of different plant parts revealed that the accumulation of ononitol in leaves was linearly related to stem m60MT activity during drought stress, indicating that m6OMT may control the in vivo biosynthetic rate of this cyclitol. The availability of myo-inositol, required for enhanced rates of ononitol synthesis by m60MT, increased during the stress experiment, while the capacity to synthezise AdoMet by S-adenosyl-L-methionine synthetase (SMS, EC 2.5.1.6) decreased. However the high capacity for degradation of S-adenosyl-L-homocysteine (AdoHcy; a potent competitive inhibitor of m60MT) by the enzyme S-adenosyl-L-**homocysteine hydrolase** (SHH, EC 3.3.1.1) provided favourable conditions for ononitol biosynthesis during the whole stress treatment.

L22 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2003 THOMSON ISI
ACCESSION NUMBER: 93:736580 SCISEARCH
THE GENUINE ARTICLE: ML589
TITLE: Z-4',5'-DIDEHYDRO-5'-DEOXY-5'-FLUOROADENOSINE (MDL-28,842), AN IRREVERSIBLE INHIBITOR OF S-ADENOSYLHOMOCYSTEINE HYDROLASE, SUPPRESSES PROLIFERATION OF CULTURED KERATINOCYTES AND SQUAMOUS CARCINOMA CELL-LINES
AUTHOR: PALLER A S (Reprint); ARNSMEIER S L; CLARK S H; MIRKIN B L
CORPORATE SOURCE: NORTHWESTERN UNIV, SCH MED, CHILDRENS MEM INST EDUC & RES, DEPT PEDIAT, CHICAGO, IL, 60611; NORTHWESTERN UNIV, SCH MED, CHILDRENS MEM INST EDUC & RES, DEPT DERMATOL, CHICAGO, IL, 60611; NORTHWESTERN UNIV, SCH MED, CHILDRENS MEM INST EDUC & RES, DEPT PHARMACOL, CHICAGO, IL, 60611
COUNTRY OF AUTHOR: USA
SOURCE: CANCER RESEARCH, (15 DEC 1993) Vol. 53, No. 24, pp. 6058-6060.
ISSN: 0008-5472.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 19

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB **S-Adenosylmethionine**-dependent transmethylation reactions are required for many critical pathways in human cells. The

enzyme S-adenosylhomocysteine hydrolase converts S-adenosylhomocysteine, a potent endogenous inhibitor of **S-adenosylmethionine** -mediated **methyltransferase** reactions, to adenosine and L-homocysteine. The effects of the inhibitor of S-adenosylhomocysteine hydrolase, Z-4',5'-didehydro-5'-deoxy-5'-fluoro-adenosine (MDL 28,842), on the growth -of cultured keratinocytes and cutaneous squamous cell carcinoma lines were investigated. MDL 28,842 suppressed the proliferation of all cells in a dose-dependent manner, and significantly increased keratinocyte differentiation at a concentration of 1 muM. Following incubation with MDL 28,842, the methylation indices (ratio of **S-adenosylmethionine**/S-adenosylhomocysteine) of undifferentiated keratinocytes and squamous cell carcinoma lines were significantly decreased.

These data demonstrate that the inhibitory effect of MDL 28,842 on squamous carcinoma cells and keratinocyte proliferation may result directly from inhibition of S-adenosylhomocysteine hydrolase activity. The antiproliferative activity of MDL 28,842 against squamous carcinoma cells and keratinocytes suggests a potential role for MDL 28,842 as a novel therapeutic agent for neoplastic and hyperproliferative disorders of the skin.

L22 ANSWER 5 OF 6 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 81096248 MEDLINE
DOCUMENT NUMBER: 81096248 PubMed ID: 7452498
TITLE: Decreased cerebral catabolism of [3H]histamine in vivo after **S-adenosylmethionine** administration.
AUTHOR: Schatz R A; Stramentinoli G; Sellinger O Z
CONTRACT NUMBER: 06294
SOURCE: JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, (1981 Jan) 216 (1) 118-24.
Journal code: 0376362. ISSN: 0022-3565.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198103
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19900316
Entered Medline: 19810317

AB Administration of S-adenosyl-L-methionine (SAM) (200 mg/kg) to adult mice significantly elevated its cerebral levels while the steady-state levels of histamine (HA) and S-adenosyl-L-homocysteine remained unaltered. [3H]HA (1 microCi/10 microliters) was injected intraventricularly (i.vt.) 20 sec, 2, 5, 10 or 20 min prior to sacrifice (1 hr after SAM) and brains were **analyzed** for [3H]HA, [3H]methylhistamine (MeHA) and [3H]methylimidazoleacetic acid. Brains of SAM-treated mice contained more [3H]HA than vehicle-treated controls at 20 sec, 2, 5 and 10 min (22, 35, 52 and 25%, respectively). [3H]MeHA levels were lower than controls at 20 sec, but higher at 2 and 5 min. Fifteen minutes after i.vt. [3H]histidine, brains of SAM-treated mice contained 47% more [3H]HA and 39% more [3H]MeHA (compared to controls) while [3H]histidine and [3H]methylimidazoleacetic acid levels remained unchanged. SAM treatment had no effect on the activity of cerebral histamine-N-**methyltransferase**, S-adenosyl-L-**homocysteine** **hydrolase** and monoamine oxidase type A (substrate 5-hydroxytryptamine) when tested in vitro, while monoamine oxidase B (substrate phenylethylamine) activity was significantly decreased. In vitro, SAM had no effect on monoamine oxidase A or B. The findings demonstrate that, unexpectedly, the rate of catabolism of HA to MeHA is significantly decelerated in brains containing elevated levels of SAM.

L22 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1979:470528 CAPLUS
DOCUMENT NUMBER: 91:70528
TITLE: New method for the separation of adenosine nucleoside derivatives by high-performance liquid chromatography. Application to the **determination** of S-methyl-L-**homocysteine** **hydrolase** from rat liver
AUTHOR(S): Chabannes, B. E.; Bidard, J. N.; Sarda, N. N.;

Cronenberger, L. A.
CORPORATE SOURCE: Serv. Chim. Biol., Inst. Natl. Sci. Appl., Villeurbanne, 69621, Fr.
SOURCE: Journal of Chromatography (1979), 170(2), 430-6
CODEN: JOCRAM; ISSN: 0021-9673
DOCUMENT TYPE: Journal
LANGUAGE: French
AB Conditions were **detd**. for sepn. of S-adenosyl-L-homocysteine (I) by paired-ion chromatog. on .mu.-Bondapak C18 in aq. MeOH contg. heptanesulfonic acid, with an absorbance detector operating at 254 nm. The facile sepn. of I from related compds. permitted **detn**. of I hydrolase by **measuring** I formation from adenosine and homocysteine; this reaction need not be coupled to a 2nd enzymic reaction as does the hydrolytic reaction on which previous assays were based. Catechol O-**methyltransferase** was also **measured** by I **detn**. in reaction mixts. after incubation of enzyme and **S -adenosylmethionine** with dopamine or noradrenaline.

=> log y